INTRODUCTION

Sinus of Valsalva aneurysm (SVA) is a very infrequent cardiac anomaly (0.14-0.96% of cases of open-heart surgery). Although the cause may be acquired, most cases are congenital anomalies arising from a defect of the aortic media. SVA usually remains silent until it ruptures, although it sometimes produces different clinical manifestations, such as obstruction of the right ventricular outflow tract, aortic regurgitation, rhythm disorders and, more rarely, myocardial ischemia or necrosis. We present a case of congenital right SVA that presented as inferior acute myocardial infarction (AMI).

CLINICAL CASE

The patient was a 40-year-old white male who smoked, but had no other coronary risk factors. In childhood he had been advised of the presence of a systolic murmur, but until the day of admission it had been asymptomatic and had not been studied. The patient came to the hospital with prolonged chest pain that had appeared suddenly while at rest. In the emergency room he was diagnosed as inferior AMI with clinical and electrocardiographic criteria, which later was confirmed by the demonstration of enzyme elevation (CPK 3495, MB 362). The clinical evolution was uncomplicated and 7 days after admission the patient was transferred to our service for complete study. In the physical examination his blood pressure was 100/60 mm Hg and he had a precordial systolic ejection murmur, grade 3/6. The ECG showed sinus rhythm, 65 beats/min, with a QS pattern and negative T waves in leads III and aVF. The chest X-ray exam was normal. Transthoracic echocardiography showed a tricuspid aortic valve and a partially thrombosed anterior sinus of Valsalva aneurysm. The left ventricle was dilated (end-diastolic diameter

Aneurysmal dilatation of one or more of the sinuses of Valsalva (SVA) is a rare cause of coronary insufficiency. We describe one case of unruptured and partially thrombosed right sinus of Valsalva aneurysm of which the first sign was acute inferior myocardial infarction in a 40-year-old man while reviewing the literature, we found 44 reported cases of sinus of Valsalva aneurysm, complicated by myocardial ischemia or infarction. In 28 cases the left coronary sinus was involved, in 12 cases the right one, and in 4 cases both of them. Myocardial ischemia is a potentially ominous prognostic sign in SVA patients. The poor outcome with conservative treatment leads us to consider the patient for emergency surgical therapy.

Key words: Aneurysm. Valsalva. Ischemia. Myocardial infarction.
Discussed.
less frequently to mechanical deformation of the os-
tium and/or proximal compression.8

In our case, the SVA was probably congenital (pos-
sible acquired causes were excluded) and the first cli-
nical manifestation was inferior AMI. In the interna-
tional literature, only 44 cases of SVA accompanied
by myocardial ischemia or necrosis have been repor-
ted. Of them, 28 involved the left sinus of Valsalva, 12
the right sinus, and 4 both. AMI was the first clinical
manifestation in 14 of the 28 cases of left SVA, but
only in 3 of the 12 right SVA (Table 1). Our patient,
consequently, is the fourth case in which AMI was the
first manifestation of an unruptured right SVA and
the first described in the Spanish literature.

Myocardial ischemia secondary to SVA can be a
sign of poor prognosis. As indicated by Faillace et al.,7
aneurysmal dilatation may occur very rapidly, and
early diagnosis and surgical treatment is mandatory.
The surgical technique depends on the anatomical
constraints and ranges from a simple patch closing the
aneurysmal orifice9 to complete reconstruction of the
aortic root. The prognosis seems to improve if aortic
valve replacement and aortocoronary bypass can be
avoided.10 After surgical repair, the prognosis is
usually good and the risk of recurrence is rare.

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I et al. Aneurysm of the left aortic sinus causing co-
ronary compression and unstable angina: successful repair by iso-

<table>
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<th>Mechanism</th>
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<tr>
<td>Chipps, 1941</td>
<td>Left</td>
<td>Compression of LCT, DA and CX</td>
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<td>Bulkley, 1975</td>
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<td>Aol. Sudden death</td>
</tr>
<tr>
<td>Cupo, 1981</td>
<td>Left</td>
<td>Unknown</td>
<td>NCS also aneurysmal</td>
</tr>
<tr>
<td>Hijamuta, 1983</td>
<td>Left</td>
<td>Compression of LCT and DA</td>
<td>Aol</td>
</tr>
<tr>
<td>Brandt, 1985</td>
<td>Left</td>
<td>Compression of LCT, DA and CX</td>
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<td>Ishide, 1985</td>
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<td>Compression of RC</td>
<td>Moderate Aol. Weber-Christian disease</td>
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<td>Faillace, 1985</td>
<td>Left</td>
<td>Compression of LCT</td>
<td>Aol. Rupture into LV</td>
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<td>Ince, 1986</td>
<td>Left</td>
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<td>Nakano, 1986</td>
<td>Left</td>
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<td>Koike, 1991</td>
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<tr>
<td>Wiemer, 1996</td>
<td>Left</td>
<td>Compression of DA and CX</td>
<td>Congenital AoS. IE</td>
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</tbody>
</table>

ALE indicates acute lung edema; AoI, aortic regurgitation; AoS, aortic stenosis; AoV, aortic valve; AoVR, aortic valve replacement; CX, circumflex artery; DA, left descending anterior artery; IE, infectious endocarditis; LA, left atrium; LCT, left coronary trunk; LV, left ventricle; MI, mitral regurgitation; MS, mitral stenosis; NCS, non-coronary sinus; RC, right coronary artery; RV, right ventricle.